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Letter to the Editor

Regarding van den Bedem, Schutte, van der Helm, and Simonsz: Mechanical properties and functional importance of pulley bands or 'Faisseaux Tendineux' ☆

In their paper entitled "Mechanical properties and functional importance of pulley bands or 'Faisseaux Tendineux', van den Bedem et al. (*Vision Research* 45 (2005) 2710–2714) argue that mechanical properties of connective tissues they define as "pulley bands" are unsuited for stabilization of rectus extraocular muscles (EOMs) (van den Bedem, Schutte, van der Helm, & Simonsz, 2005). Such claims seem likely to confuse rather than clarify the issue.

van den Bedem et al.'s quotation from Tenon describes lateral bending of the lateral rectus (LR) path. Tenon surmised that during LR contraction, fascia would inflect the LR path laterally in the manner of a pulley, as also noted by Sappey (Sappey, 1888, 2001). While magnetic resonance imaging (MRI) of living orbits confirms as physiologic such lateral inflection of horizontal rectus EOM path (Demer, Oh, & Poukens, 2000, Demer, 2005), this is not the pulley action proposed in the active pulley hypothesis (APH), which rather is resistance to EOM sideslip (Demer, 2004, 2005; Demer et al., 2000).

Studies not cited by van den Bedem et al. suggest that orbitally coupled connective tissues are responsible for rectus EOM resistance to sideslip, and for sharp EOM path inflections. Initial evidence for orbitally coupled pulleys was the MRI observation that posterior EOM paths are little altered by large surgical transpositions of their insertions (Clark, Rosenbaum, & Demer, 1999; Miller, Demer, & Rosenbaum, 1993). This finding, and persistence of EOM path inflections even after globe enucleation (Detorakis, Engstrom, Straatsma, & Demer, 2003), have ruled out musculo-global connections as the basis of EOM path stability. MRI has subsequently demonstrated in secondary (Clark, Miller, & Demer, 2000) and tertiary (Kono, Clark, & Demer, 2002a) gaze positions large anteroposterior shifts in path inflections during EOM contraction and relaxation (Kono et al., 2002a).

Orbitally-stabilized pulleys change EOM pulling direction as eye position changes, fundamentally influencing neural control of eye movements (Angelaki, 2003; Angelaki & Hess, 2004; Crawford, Martinez-Trujillo, & Kleier, 2003; Demer, 2004; Miller, 1989; Miller & Demer, 1995; Raphan, 1997; Raphan, 1998). Half angle dependence of ocular rotational velocity axis on eye position is equivalent to Listing's Law (LL) (Tweed & Vilis, 1990). Neurophysiological observations are consistent with a mechanical basis for LL: motoneurons innervating vertical rectus and oblique EOMs do not encode LL torsion during pursuit (Ghasia & Angelaki, 2005), while direct abducens nerve stimulation evokes saccades conforming to LL (Klier, Meng, & Angelaki, 2005). Large shifts in rectus pulley positions underlying LL were predicted by the APH (Demer et al., 2000), and confirmed by MRI (Kono et al., 2002a). van den Bedem et al. suggest that the lateral rectus pulley suspension is too stiff to permit large pulley shifts. That implication of van den Bedem et al. seems irreconcilable with the in vivo MRI observation that the four human rectus pulleys actually do shift (Kono et al., 2002a).

van den Bedem et al. have not considered the primary sources describing modern anatomy of orbital connective tissue system. Pulleys that inflect rectus EOM paths have been histologically characterized in detail (Demer, 2000; Demer, Miller, Poukens, Vinters, & Glasgow, 1995; Demer, Poukens, Miller, & Micevych, 1997; Kono, Poukens, & Demer, 2002b). Modern studies have avoided artifacts that probably caused pulley function to be overlooked earlier. Recent work has incorporated serial sectioning of intact orbits subjected previously to MRI enabling compensation of shrinkage and other artifacts of processing. Comparison of dissected tissues with serially sectioned orbits not internally disturbed has revealed that dissection distorts tissue relationships normally maintained by balanced elastic tensions (Demer, 2004). The surgically exenterated, partial, unfixed, and orbits studied by van den Bedem et al. would have been subject to marked alterations of soft tissue relationships, particularly near the pulley system attachment to bone (Kono et al., 2002b). Lysis from bone is traumatic, making it virtually impossible to later identify tissues for mechanical measurements. While Fig. 2 of van den Bedem et al. illustrates an axial histological section of an orbit published earlier (Koornneef, 1977), it cannot assure that the experimenters were measuring the origin of the intact pulley band. Likelihood of tissue damage is suggested by the grossly visible orbital fat in Video 1 of van den Bedem et al.

van den Bedem et al.'s removal exenteration allowed the elastic tissues to contract inward into an unsupported

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mass. As is suggested by Video 1, the LR EOM stump had apparently contracted anteriorly into the connective tissues. Video 2 of van den Bedem et al. shows counter-traction applied with forceps to lateral tissues, while a spring scale attached to another site was displaced. From this imprecisely controlled arrangement, van den Bedem et al. concluded that the pulley suspension has a nonlinear characteristic, with almost no stiffness for displacements estimated as up to 10 mm, and marked stiffness for greater displacements. This experiment and its interpretation are fundamentally flawed by failure to consider rest length of the connective tissues involved, and failure to measure actual displacements imposed. Video 2 indicates that tissues were relaxed to redundancy at initial stretch, which would be interpreted as zero initial stiffness. There was then a range of elasticity over some further displacement, followed by almost complete resistance to final displacement. van den Bedem et al. do not provide quantitative stiffness values for their measurements, and cannot know if initial tissue length or preload were physiologic. The range of modest elasticity between the slack and "leash" zones might have represented the physiologic range of extensibility for the pulley suspension. The experiment is at most inconclusive, and certainly does not disprove the APH.

van den Bedem et al. presented a patient with Crouzon's syndrome and the "V" pattern exotropia typical of the abnormal rectus EOM paths illustrated in Fig. 3 of their paper. An LR path inferior to that of the medial rectus gives the LR an abnormal infraducting action consistent with the "V" pattern (Demer, 2002, 2003). It is difficult to accept the statement of van den Bedem et al. that the eye movements of this patient illustrated in Video 3 violated LL; such kinematics cannot be determined from casual inspection of ocular rotations, and are certainly not obvious from the video. Quantitative three-dimensional eye movement recording is required to support assertions concerning LL kinematics. But consider: if the patient violates LL, how could ocular motility be called normal?

van den Bedem et al. make the unwarranted inference that shallow orbits in Crouzon syndrome preclude stabilization of the pulleys by orbital bones. Normal orbits have no direct connection of the superior and inferior rectus EOMs to the bones, but instead indirect connections to the medial and lateral orbit (Kono et al., 2002b). Even if parts of the globe were anterior to some orbital bones, the soft tissues and slightly more remote bones could stabilize the rectus pulleys. Pathologic connective tissues do result in rectus EOM sideslip and consequent strabismus (Demer, Kono, Wright, Oh, & Clark, 2002; Oh, Clark, Velez, Rosenbaum, & Demer, 2002). If one compares the middle with the lower panels, considerable lateral sideslip of the right IR muscle in right gaze is evident in Fig. 3 of van den Bedem et al. IR pulley instability may have contributed to incomitant strabismus in the patient. Fig. 3 also shows globe translation with lateral gaze shift, apparently 3–4 mm based on the disappearance of globe cross section of the adducting eye. Such translation is much larger than normal (Clark et al., 2000). The Crouzon's case hardly supports the conclusion that ocular motility is normal when the orbital supporting tissues are grossly abnormal.

van den Bedem et al. stated "that no sideways displacement of the (left) inferior rectus muscle occurred on left and right gaze (Video 4), although the (thin) inferior oblique muscle had been severed from the inferior rectus muscle and the orbital floor was lacking." Intrasurgical Video 4 is not convincing on this point, since all that is visible in the video beyond the inferior conjunctiva is sutures presumably attached to the severed inferior rectus (IR) tendon. Contrary to assertions of van den Bedem et al., suture traction on the IR tendon plainly shifted the tendon transversely. However, possible shift of the deeper IR pulley and belly cannot be determined from Video 4.

van den Bedem et al. imply unavailability of histological data for the LR pulley band. However, Kono et al. published quantitative analysis of collagen and elastin in orbital connective tissues of four whole human orbits, emphasizing the pulleys and their suspensions (Kono et al., 2002b). A ring-sling computational model of horizontal rectus pulleys based on the data of Kono et al. produces pulley behavior and simulated EOM tensions consistent with the APH and with available physiological data (Vijayaraghavan & Demer, 2005).

Quantitative anatomic and physiologic data support the APH proposition that EOM paths are constrained by actively controlled connective tissue pulleys. Further investigation of the mechanical properties of the orbital tissues would be welcome.

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